

# ELECTROCARDIOGRAPHIC CHANGES DURING PROFOUND HYPOTHERMIA

BY

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In an endeavour to develop a safe and easy technique for operating on the open heart, profound hypothermia was induced in dogs by means of an extra-corporeal circulation with pump oxygenator and heat exchanger until the temperature in the pharynx reached 15° C. The circulation was then arrested completely for up to 45 minutes before warming was commenced. The success of these experiments lead to the use of the method in fourteen patients. During the cycle of cooling, complete circulatory arrest, and warming, the changes in the electrical activity of the myocardium were found to follow a regular pattern which is described in this paper.

## METHODS

Hypothermia was induced by means of a low-flow partial cardio-pulmonary bypass through a modified de Wall bubble oxygenator (De Wall *et al.*, 1956; Cooley *et al.*, 1958) and a heat exchanger (Brown *et al.*, 1958) the blood being taken from the right atrium and returned to the patient through a cannula placed in the external iliac artery. If the atrial septum was intact the left atrium was decompressed by draining its blood into the venous reservoir of the machine whenever cardiac action was impaired.

Anæsthesia was induced with thiopentone, and maintained with intratracheal nitrous oxide and oxygen, with d-tubocurarine to facilitate the control of respiration. During the bypass no anæsthetic agent was given, pure oxygen was administered through the endo-tracheal tube, and a mixture of 95 per cent oxygen and 5 per cent carbon dioxide was used in the bubble oxygenator.

Temperatures were measured with glass thermometers in the pharynx and in the pericardial cavity adjacent to the myocardium. The lowest pharyngeal temperatures varied between 13° C and 18° C. and the lowest temperatures measured in the pericardial cavity between 5° and 14° C. (Fig. 1).

When the pharyngeal temperature was in the vicinity of 15° C. circulatory arrest was obtained by turning off the pump oxygenator and occluding the venæ cavæ. The longest period of circulatory arrest was 53 minutes, and the shortest 12 minutes.

A continuous electrocardiographic record was made throughout the phases of cooling, arrest, and warming in 11 cases using a Sanborn twin viso direct writer, running at paper speeds of either one, ten, or twenty-five millimetres per second. Incomplete records were obtained in the other three cases using an N.E.P. photographic recorder.

Of the fourteen patients, eight had atrial septal defects of the secundum type and two of these had associated partial anomalous pulmonary venous drainage; one patient had an atrial septal defect of the 'primum' type and a separate secundum defect. Two patients had Fallot's tetralogy, and one of these also had an absent left pulmonary artery. Of the remaining three, one had rheumatic

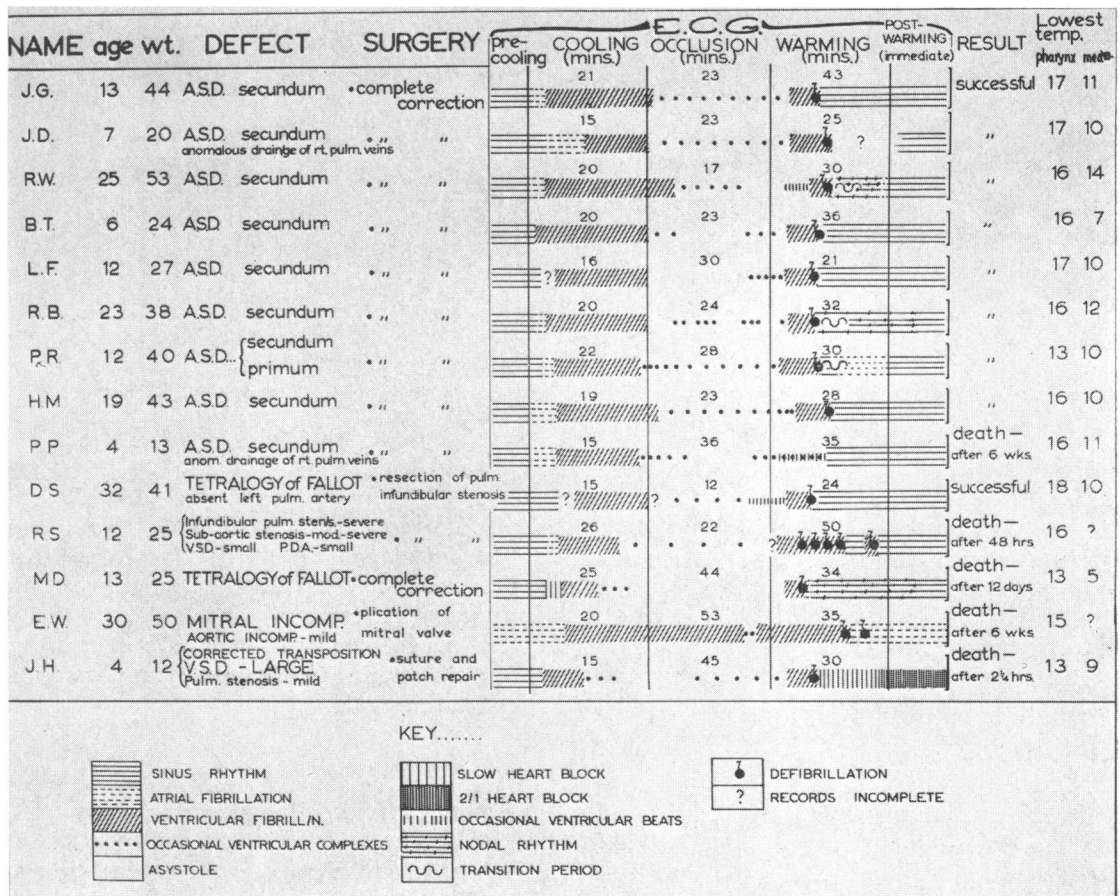


FIG. 1.—Table of electrocardiographic changes with clinical and bypass data in 14 patients subjected to deep hypothermia.

mitral regurgitation, one a large ventricular septal defect with corrected transposition, and the third had a complicated defect consisting of severe infundibular stenosis, a ventricular septal defect 3 mm. in diameter, a patent ductus arteriosus, and sub-aortic stenosis (Fig. 1).

The patient with mitral regurgitation was the only one receiving digitalis before operation. Quinidine was not used.

#### OBSERVATIONS

In the fourteen patients subjected to deep hypothermia by the technique described above, the electrocardiographic features in standard lead II were as follows.

Sinus rhythm was present before cooling in every tracing except one which showed atrial fibrillation. Within a minute or two of the beginning of cooling, the sinus rhythm slowed and usually changed to atrial fibrillation, but in a few cases there were periods of nodal rhythm. Right atrial activity at this stage was influenced by the manipulation of the cannulae which were tied into this chamber. Tall and broad T waves and J waves with inverted T waves (Emslie-Smith *et al.*, 1959) were often seen.

Ventricular fibrillation occurred within the first eight minutes of cooling. The frequency and amplitude of the fibrillation waves diminished as cooling progressed (Fig. 2). In five cases the

ventricular fibrillation changed to an irregular or regular ventricular rhythm several minutes before circulatory arrest (Fig. 1).

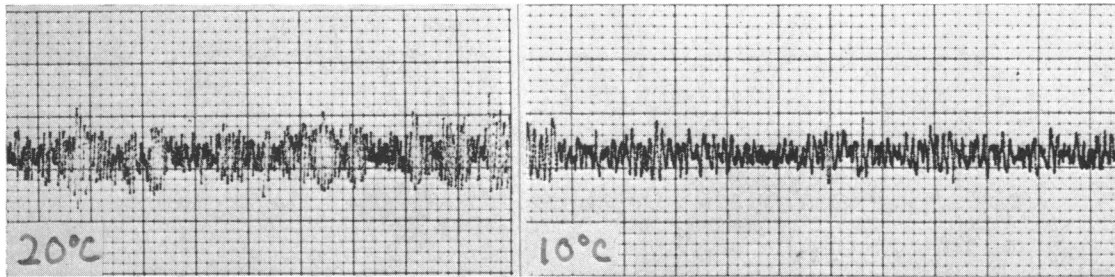


FIG. 2.—Shows the decrease in amplitude of fibrillation waves during cooling. The first section was recorded when the mediastinal temperature was at 20° C. and the second when it was 10° C. Paper speed 1 millimetre per second.

During complete circulatory arrest when the pharyngeal temperature was approximately 15° C. all ventricular activity usually ceased and cardiac asystole supervened. This was interrupted at times by occasional ventricular complexes which were sometimes initiated by mechanical stimulation of the heart. The electrical complexes were at times accompanied by extremely slow visible muscular contractions. Atrial waves were also occasionally observed (Fig. 3).

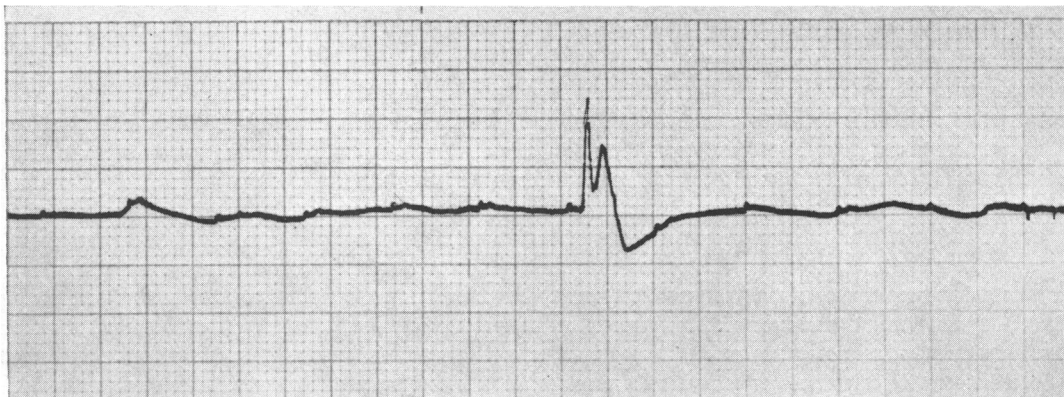


FIG. 3.—Asystole interrupted by small regular atrial deflections and a single ventricular complex. Paper speed 10 millimetres per second.

Within three minutes of the commencement of warming, at approximately 20° C. pharyngeal, the ventricular complexes, if present, were replaced by ventricular fibrillation, except in one case in which normal rhythm returned spontaneously. The ventricular fibrillation waves increased in frequency and amplitude until electrical defibrillation was carried out (Fig. 4).

Defibrillation was usually successful after one or two shocks with the defibrillator when the pericardial temperature had reached 30° C. Only once was there difficulty in defibrillation, and this occurred in a case of unrecognized sub-aortic stenosis.

After defibrillation bizarre ventricular complexes occurred for short periods. Sometimes a short burst of atrial fibrillation or of nodal rhythm preceded the onset of normal sinus rhythm. The return of sinus rhythm often seemed to be related to the removal of the atrial cannulæ.

The electrocardiographic standard lead II at the end of operation showed no significant QRS, S-T segment, or T wave changes from that before operation.

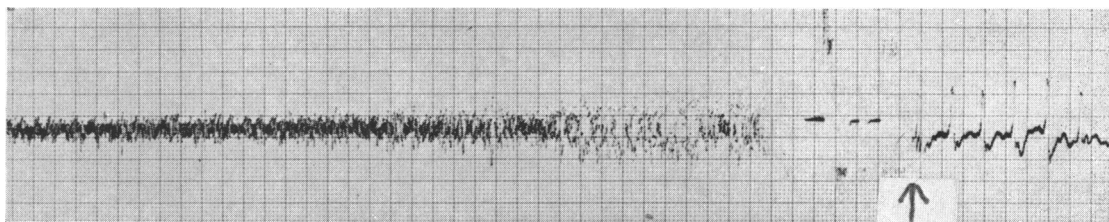


FIG. 4.—Shows the increase in amplitude of the fibrillation waves prior to defibrillation (indicated by arrow). The cardiograph was turned off during defibrillation. Paper speed 1 millimetre per second before and 10 millimetres per second after defibrillation.

#### DISCUSSION

The broad pattern of the electrocardiographic changes is similar to that described by Drew (1961) except that ventricular fibrillation was encountered during the warming phase in all cases except one, whereas Drew met it in only one-third of his. Nine of these fourteen patients were under the age of 14 which contrasts with Drew's statement that defibrillation is rarely necessary in children. An explanation could lie in Drew's higher perfusion flow rates—he maintained a mean blood pressure of 70–80 mm. Hg compared with approximately 40 mm. Hg in this series—suggesting that decreased coronary flow is one of the factors predisposing to ventricular fibrillation. On the other hand Simpson *et al.* (1960) met with ventricular fibrillation in each of six cases where the perfusion pressure was about 70 mm. Hg, so that cold itself may be the more important factor.

It is noteworthy that ventricular fibrillation can change “spontaneously” to slow regular ventricular complexes during the late cooling or occlusion phase. No satisfactory explanation can be advanced for this.

The exact levels of potassium and calcium ions were not known in these cases. The pH was in the region of 7.2 during the cold phase but after the return to normal temperature it was again over 7.4. The arterial  $p\text{CO}_2$  was not measured. These parameters are said to be important in the limitation of arrhythmias (Cooper, 1961).

#### SUMMARY

During the induction of deep hypothermia in 14 cases by the technique described, normal sinus rhythm first slowed, then changed to atrial fibrillation, and finally to ventricular fibrillation.

During a period of complete circulatory arrest at a pharyngeal temperature of between 13° C. and 18° C., ventricular fibrillation was usually replaced by asystole and this was often interrupted by regular or irregular ventricular electrical complexes and occasionally by ventricular contractions.

During the warming phase, ventricular fibrillation usually reappeared.

Electrical defibrillation during warming was followed by bizarre ventricular complexes, atrial fibrillation, or nodal rhythm, before the return of normal sinus rhythm.

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